LUNG CANCER AND EXPOSURE TO TOBACCO SMOKE IN THE HOUSEHOLD

DWIGHT T. JANERICH, D.D.S., M.P.H., W. DOUGLAS THOMPSON, PH.D., LUIS R. VARELA, M.D., PH.D.,* PETER GREENWALD, M.D., DR.P.H., SHERRY CHOROST, M.S., CATHY TUCCI, B.S., MUHAMMAD B. ZAMAN, M.D., MYRON R. MELAMED, M.D., MAUREEN KIELY, R.N., AND MARTIN F. MCKNEALLY, M.D.

Abstract *Background.* The relation between passive smoking and lung cancer is of great public health importance. Some previous studies have suggested that exposure to environmental tobacco smoke in the household can cause lung cancer, but others have found no effect. Smoking by the spouse has been the most commonly used measure of this exposure.

Methods. In order to determine whether lung cancer is associated with exposure to tobacco smoke within the household, we conducted a population-based case–control study of 191 patients with histologically confirmed primary lung cancer who had never smoked and an equal number of persons without lung cancer who had never smoked. Lifetime residential histories including information on exposure to environmental tobacco smoke were compiled and analyzed. Exposure was measured in terms of "smoker-years," determined by multiplying the number of years in each residence by the number of smokers in the household.

Results. Household exposure to 25 or more smoker-years during childhood and adolescence doubled the risk of lung cancer (odds ratio, 2.07; 95 percent confidence interval, 1.16 to 3.68). Approximately 15 percent of the control subjects who had never smoked reported this level of exposure. Household exposure of less than 25 smoker-years during childhood and adolescence did not increase the risk of lung cancer. Exposure to a spouse's smoking, which constituted less than one third of total household exposure on average, was not associated with an increase in risk.

Conclusions. The possibility of recall bias and other methodologic problems may influence the results of case–control studies of environmental tobacco smoke. Nonetheless, our findings regarding exposure during early life suggest that approximately 17 percent of lung cancers among nonsmokers can be attributed to high levels of exposure to cigarette smoke during childhood and adolescence. (N Engl J Med 1990; 323:632-6.)

THE 1972 Surgeon General's report dealt with the health consequences of passive smoking or environmental tobacco smoke for the first time.[1] In 1986 the entire report was devoted to the issue; it concluded that "involuntary smoking is a cause of disease including lung cancer in healthy non-smokers."[2] More than a dozen epidemiologic studies have assessed the relation between passive smoking and lung cancer.[3-17] The findings have ranged from no detectable increase in risk[10,17] to a moderate (about twofold), statistically significant increase.[4-7] Most studies have found only small elevations in risk, which are frequently not statistically significant.[3,8,9] In a meta-analysis of all the available studies in 1986, Wald et al. found a slightly increased risk of lung cancer associated with environmental tobacco smoke.[18]

We undertook the current study in an attempt to clarify further the role of passive smoking in causing lung cancer. In this report we discuss exposure to tobacco smoke in the household as a possible cause of lung cancer among nonsmokers.

METHODS

We conducted a population-based, individually matched case–control study in New York State from 1982 to 1985. The cases were drawn from seven Standard Metropolitan Statistical Areas (Buffalo, Rochester, Syracuse, Utica–Rome, Albany–Schenectady–Troy, Binghamton, and greater New York, excluding the five boroughs of New York City). This geographic area comprises 23 counties, with approximately 125 diagnostic or treatment facilities, and a population base of nearly 10 million people. A special system for the rapid ascertainment of cases of lung cancer was established in these 125 facilities so that patients could be identified and enrolled as soon after diagnosis as possible. All new cases of lung cancer (diagnosed clinically, histologically, or both) were regularly identified at the participating hospitals. The New York State Cancer Registry was checked routinely to identify any cases that might have been missed by the hospital-based reporting system.

Information on smoking was initially obtained from the patients' medical records. All the case patients reported as having never smoked or as former smokers or whose smoking history was unknown were contacted by telephone, and their smoking status was confirmed. To be included as a "case" in the study, a patient had to reside in the 23-county area, be between 20 and 80 years of age, never have smoked more than 100 cigarettes (nonsmokers) or have smoked at some time but not have smoked more than 100 cigarettes in the 10 years before diagnosis (former smokers), and have been given a diagnosis of primary lung cancer between July 1, 1982, and December 31, 1984, that was confirmed on reexamination of the pathological specimens and clinical records. Slides or blocks of tissue were available for all but five of the case patients. All materials were reviewed by investigators who were blinded with respect to the patient's initial diagnosis, smoking history, and other risk factors. Interviews were conducted with 76 percent of the eligible patients or their closest available relatives or friends (surrogates).

Control subjects were individually matched to the patients and were selected by screening the files of the New York State Department of Motor Vehicles. This source of controls was considered appropriate since it was population-based and provided most of the information necessary to perform the matching. A list of potential control subjects for each case patient was selected on the basis of age (within five years), sex, and county of residence. Potential control subjects were contacted by telephone. The first eligible subject who was found to match the case patient in terms of smoking history (nonsmoker or former smoker) and who agreed to participate was enrolled in the study. An additional matching variable considered at the time of data collection was the type of interview — i.e., direct interview with the patient or control subject versus interview with a surrogate respondent. When a surrogate case patient had to be interviewed, we also interviewed a surrogate for his or her matched

From the Department of Epidemiology and Public Health, Yale University School of Medicine, New Haven, Conn. (D.T.J.); the University of Southern Maine, Portland (W.D.T.); the Population Council of Mexico, Mexico City (L.V.R.); the National Cancer Institute, Washington, D.C. (P.G.); the New York State Department of Health, Albany (S.C., C.T.); the Sloan-Kettering Memorial Institute, New York (M.B.Z., M.R.M.); and Albany Medical College, Albany, N.Y. (M.K., M.F.M.). Address reprint requests to Dr. Janerich at the Department of Epidemiology and Public Health, Yale University School of Medicine, 60 College St. (LEPH 105), New Haven, CT 06510.

Supported in part by grants (PO1 CA 42101 and RO1 CA 32068) from the National Institutes of Health.

*Deceased.

2023382397

Source: https://www.industrydocuments.ucsf.edu/docs/zkyx0000

control, even when the control subject was available and willing to be interviewed. Further information on the methods used in the study is available elsewhere.[19]

Data were collected for 439 case–control pairs. Of these, 242 pairs were former smokers and 197 pairs had never smoked. Separating the residual effects of direct smoking from those of passive smoking among former smokers involves more complex analytic and interpretational issues than does an examination of the effects of passive smoking in those who have never smoked. This report is therefore limited to persons who never smoked. Six of the 197 pairs who had never smoked were mismatched in terms of the type of interview (direct vs. surrogate) and have therefore been excluded. Thus, the analyses reported here were based on 191 matched case–control pairs. A total of 129 pairs were interviewed directly, and surrogates were interviewed for 62.

All information was collected during a face-to-face interview with use of a precoded questionnaire. Case patients and control subjects were interviewed in exactly the same fashion, and except for items concerning the clinical aspects of the current medical condition, both groups answered the same questions.

Information about smoking in the household was collected separately for each residence in which the subject had lived for one year or more, up to a maximum of 12 residences. The number of "smoker-years" of exposure was calculated by multiplying the number of years the subject lived in each residence by the number of smokers (including the spouse) in that residence. The products for all residences were summed.

Smoking by the spouse was also recorded separately from that by other household members in a subsequent section of the questionnaire. The information consisted of the number of years the spouse had smoked while living with the case patient or control subject and the number of cigarettes smoked per day. Smoker-years of exposure from the spouse's smoking were calculated in the same manner as for the entire household. Pack-years of exposure from the spouse were calculated by multiplying the number of packs smoked per day by the number of years that the spouse smoked while living with the subject. If the subject had been married to more than one smoker, then the numbers of smoker-years and pack-years of exposure for all spouses were summed.

The questionnaire also included sections on exposure to environmental tobacco smoke in the workplace and in social settings outside the home. The format for these questions differed from that used to collect data on exposure in the household. The summary results of this analysis are presented here; detailed findings are available elsewhere.[19]

Statistical techniques appropriate for the analysis of individually matched case–control studies were used.[20] For clarity of presentation, percentages were tabulated for case patients and control subjects separately, rather than for matched pairs. However, odds ratios were computed on the basis of the matched pairs. The conditional logistic-regression model was used in the multivariate analyses.[20] Comparisons of the effects of exposures that occurred during different periods of the subjects' lives were based on evaluation of differences in the magnitude of appropriate logistic-regression coefficients. For statistical testing of these differences, we used the variance–covariance matrix from the logistic-regression analyses.

Results

Smoking by spouses contributed a large proportion of lifetime exposure to environmental tobacco smoke but was not the chief source of exposure. Table 1 shows the amount of exposure to environmental tobacco smoke (expressed in smoker-years) during childhood and adolescence, during adulthood, and from the spouse for the 191 control subjects who had never smoked. There were only small differences between men and women. The spouse contributed about 30 percent of the lifetime smoker-years of exposure; the correlation coefficients for exposure from the spouse and lifetime exposure were 0.37 for men and 0.51 for women. Exposure during childhood and adolescence (<21 years of age) contributed a similar percentage of the lifetime smoker-years but correlated more closely with lifetime exposure (correlation coefficient, 0.92 for men and 0.61 for women). The average lifetime exposure was 46.6 smoker-years for men and 52.7 smoker-years for women. During adulthood, household exposure from sources other than the spouse was somewhat greater than from the spouse.

Table 1. Distribution of Smoker-Years of Exposure to Environmental Tobacco in the Household.*

Category of Exposure	Men	Women
Lifetime smoker-years, mean ±SD	46.6±53.7	52.7±42.9
Smoker-years during childhood and adolescence†		
Mean ±SD	15.4±20.6	16.1±16.2
Percent of lifetime exposure	33.1	30.6
Correlation with lifetime exposure	0.92	0.61
Smoker-years from spouse		
Mean ±SD	13.0±17.0	16.2±16.7
Percent of lifetime exposure	28.0	30.7
Correlation with lifetime exposure	0.37	0.51
Smoker-years during adulthood from sources other than spouse		
Mean ±SD	18.1±31.0	20.5±29.9
Percent of lifetime exposure	38.9	38.9
Correlation with lifetime exposure	0.91	0.83

*Based on 45 male and 146 female control subjects who had never smoked more than 100 cigarettes. Information on smoker-years of exposure from the spouse is for 45 male and 140 female control subjects. Smoker-years were calculated by multiplying the number of years the subject lived in a residence by the number of smokers in the household.

†Less than 21 years of age.

Table 2 shows the odds ratios for developing lung cancer in relation to the degree of exposure to tobacco smoke in the household for the 191 nonsmoking case–control pairs. The data are stratified by levels of exposure (measured in smoker-years) and by the periods of life when the exposure occurred. Exposures during childhood and adolescence were defined as exposures that occurred when the subjects were less than 21 years of age. Exposures during adulthood include all household exposures from 21 years of age to the time of diagnosis. Although the odds ratios for the higher exposure categories are somewhat higher than those for the lower categories, no clear dose–response relation is evident, and most of the 95 percent confidence intervals include 1.0. For exposures in childhood and adolescence, the highest level of exposure is associated with the greatest elevation in risk, and the 95 percent confidence interval excludes the null value (odds ratio, 2.07; 95 percent confidence interval, 1.16 to 3.68). For the 129 case–control pairs who were interviewed directly, the odds ratio for persons with 25 or more smoker-years of exposure in childhood and adolescence was 2.31 (95 percent confidence interval, 1.16 to 4.61).

With smoker-years during childhood and adolescence and smoker-years during adulthood treated as continuous variables and included simultaneously in a logistic model, each increment of five smoker-years of exposure during childhood and adolescence was found to increase the risk of lung cancer by 6.5 percent (95 percent confidence interval, 0.1 to 13.2). On the other hand, each additional five smoker-years of exposure

2023382398

Source: https://www.industrydocuments.ucsf.edu/docs/zkyx0000

Table 2. Relation of Smoker-Years of Exposure to Environmental Tobacco Smoke to the Risk of Lung Cancer among Persons Who Never Smoked More than 100 Cigarettes.*

No. of Smoker-Years	Case Patients	Controls	Odds Ratio (95% CI)
	no. (%)		
Childhood and adolescence†			
0	57 (29.8)	68 (35.6)	—
1–24	82 (42.9)	94 (49.2)	1.09 (0.68–1.73)
≥25	52 (27.2)	29 (15.2)	2.07 (1.16–3.68)
Adulthood			
0	44 (23.0)	39 (20.4)	—
1–24	37 (19.4)	48 (25.1)	0.64 (0.34–1.21)
25–49	46 (24.1)	50 (26.2)	0.81 (0.45–1.45)
50–74	36 (18.9)	32 (16.8)	1.00 (0.52–1.93)
≥75	28 (14.7)	22 (11.5)	1.11 (0.56–2.20)
Lifetime			
0	32 (16.8)	33 (17.3)	—
1–24	20 (10.5)	27 (14.1)	0.78 (0.36–1.67)
25–49	35 (18.3)	46 (24.1)	0.80 (0.43–1.50)
50–74	44 (23.0)	40 (20.9)	1.19 (0.63–2.27)
75–99	33 (17.3)	21 (11.0)	1.80 (0.83–3.90)
≥100	27 (14.1)	24 (12.6)	1.13 (0.56–2.28)

*Based on 191 matched case–control pairs. CI denotes confidence interval. Odds ratios are shown for a person with the exposure specified as compared with a person with no exposure (0 smoker-years). Because of rounding, percentages do not always total 100.

†Less than 21 years of age.

during adulthood were estimated to have virtually no effect on risk (95 percent confidence interval, −3.3 to +2.8 percent). The difference in the magnitude of the effect between exposure during childhood and adolescence and exposure during adulthood did not achieve statistical significance ($P = 0.12$). On the basis of the distribution of exposure levels during childhood and adolescence among the control subjects and the magnitude of the effect of early exposure, we estimate that approximately 17 percent of all lung cancers in nonsmokers can be attributed to exposure to passive smoke in the household during childhood and adolescence. On the basis of the odds ratios for the 129 case–control pairs who were interviewed directly, approximately 19 percent of lung cancer in nonsmokers appears to be attributable to exposure to environmental cigarette smoke in childhood and adolescence.

Since smoking by the spouse has been the most commonly reported measure of exposure to environmental tobacco smoke in previous studies, we examined exposure from the spouse separately, although exposure to environmental tobacco smoke from the spouse is also included in the results shown in Table 2. The odds ratios for exposure frequently differed according to the type of interview, especially for the data on exposure to a spouse's smoking. Table 3 therefore shows the results of the analyses of exposure to environmental tobacco smoke from the spouse separately for subjects interviewed directly and those for whom surrogates were interviewed. The odds ratio for the development of lung cancer for those who ever had a spouse who smoked, as compared with those who did not, was 0.93 (95 percent confidence interval, 0.55 to 1.57) for those interviewed directly. In terms of smoker-years of exposure to the spouse's smoke, the results show little effect, with an odds ratio of 1.07 for 25 or more smoker-years of exposure (95 percent confidence interval, 0.59 to 1.97). Estimates based on pack-years of exposure from the spouse were similar to those based on smoker-years. For both measures, there was little evidence of a trend according to amount of exposure among those who were exposed.

All analyses were repeated for only the case–control pairs for whom we had complete and internally consistent data for all residences and marriages. Any pair was dropped from these analyses if data were incomplete or missing for either the case patient or the control subject, leaving 113 pairs of nonsmokers. Our purpose was to ensure that our conclusions were not dependent on the particular methods we adopted to handle inconsistencies or missing items in the data set. The findings were similar to those for the entire group of 191 pairs. The odds ratio for exposure to 25 or more smoker-years in childhood and adolescence was 2.59 (95 percent confidence interval, 1.22 to 5.49).

Exposure in the workplace was measured by recording the number of smokers who worked with each study subject during his or her lifetime and the amount of time the subjects spent working with these smokers. These exposures were compared for case patients and control subjects. Estimating the odds ratio as a continuous variable for an equivalent differential of 150 person-years of exposure gave an odds ratio of 0.91 (95 percent confidence interval, 0.80 to 1.04), indicating no evidence of an adverse effect of environmental tobacco smoke in the workplace. Our assessment of smoking in social settings used an untested, semiquantitative index in which the case patient or control subject used a score of 0 through 12 to indicate his or her regular exposure to tobacco smoke in social settings during each decade of life. Cumulative lifetime reported scores ranged from nearly 0 to more than 70. The odds ratio for an increase of 20 in the cumulative score was 0.59 (95 percent confidence interval, 0.43 to 0.81). Our analysis of exposure in social settings with use of this index showed a statistically significant inverse association between environmental tobacco smoke and lung cancer.

Discussion

We found a statistically significant adverse effect of relatively high levels of exposure to environmental tobacco smoke during the early decades of life (up to the age of 21). For those who were exposed to 25 or more smoker-years during their first two decades of life, the risk of lung cancer doubled. This amount of exposure is equivalent to living with more than one smoker throughout childhood and adolescence — a high but not uncommon level of exposure. An exposure of this level was reported for approximately 15 percent of the control group. By contrast, we found no adverse effect of exposure to environmental tobacco smoke during adulthood, including exposure to a spouse who

2023382399

Source: https://www.industrydocuments.ucsf.edu/docs/zkyx0000

Table 3. Relation of Spouse's Smoking to the Risk of Lung Cancer among Persons Who Never Smoked More than 100 Cigarettes, According to Type of Interview.*

VARIABLE	TYPE OF INTERVIEW	
	DIRECT	SURROGATE
	odds ratio (95% CI)	
Ever had a spouse who smoked		
No	—	—
Yes	0.93 (0.55–1.57)	0.44 (0.19–1.02)
Smoker-years of exposure from spouse		
0	—	—
1–24	0.78 (0.41–1.50)	0.33 (0.11–1.05)
≥25	1.07 (0.59–1.97)	0.33 (0.12–0.95)
Pack-years of exposure from spouse		
0	—	—
1–24	0.71 (0.37–1.35)	0.16 (0.04–0.62)
25–49	0.98 (0.47–2.05)	0.68 (0.18–2.60)
≥50	1.10 (0.47–2.56)	0.20 (0.03–1.22)

*Based on 129 case–control pairs interviewed directly and 59 pairs for whom surrogates were interviewed. Three of the 191 pairs were excluded because for one member of the pair there was missing information about whether the subject had a spouse who smoked. Data on smoker-years of exposure were available for 129 case–control pairs with direct interviews and 56 pairs with surrogate interviews. Data on pack-years of exposure were available for 122 pairs with direct interviews and 51 pairs with surrogate interviews. CI denotes confidence interval. Odds ratios are shown for a person with the exposure specified as compared with a person with no exposure to a spouse who smoked.

smoked. Although problems of recall and other potential biases may have influenced the results, our data suggest that exposure in early life may be a limited but important contributor to the risk of lung cancer in nonsmokers. A previous study with a small number of subjects found little evidence of an elevated risk of lung cancer among nonsmokers whose parents had smoked.[14] Children of parents who smoke have been shown to be especially susceptible to respiratory problems that occur soon after exposure to environmental tobacco smoke.[2] This type of susceptibility might initiate changes that eventually lead to lung cancer when the exposed children become adults, but we know of no specific mechanism that would explain our findings.

We found no adverse effects of exposure to tobacco smoke in the workplace, although we did not have enough information about the level of exposure in the workplace to assess the precision of our measurements. The apparent protective effect of exposure in social settings is difficult to explain. During the course of this study, regulations in New York began to restrict smoking in the workplace and in social settings such as restaurants. We did not anticipate this development and cannot estimate how much the awareness of these new restrictions might have affected the responses of the study subjects or their surrogates.

Evidence is clearly mounting that tobacco smoke inhaled passively by nonsmokers is potentially carcinogenic. In a recent study, Maclure et al.[21] found elevated levels of carcinogens in the blood of passive smokers. Levels of hemoglobin adducts of 4-aminobiphenyl and adducts of 3-aminobiphenyl were significantly elevated in subjects with confirmed exposure. The validity of this finding was supported by additional evidence that showed a sharp decline in the levels of adducts among smokers who quit.[21]

At present, information on past exposure to environmental tobacco smoke can be obtained only by interview. The available biologic markers, such as cotinine, cannot be used to confirm exposure that occurred years or decades earlier. The use of interviews to obtain a lifetime history of exposure to passive smoking requires that the questionnaire be structured and the interview techniques be standardized so that all subjects are interviewed in the same way. We took steps to ensure such standardization. Two recent reports may lead to improved ways to measure lifetime exposure to environmental tobacco smoke by means of interviews.[22,23] In one of these studies, which attempted to evaluate the reliability of interview data by repeat interviews, information on exposure during childhood was found to be very reliable.[23]

It was necessary to use surrogate respondents for about one third of the interviews, usually because the patients were too ill to be interviewed. To minimize potential bias, surrogates were also interviewed for the matched control subjects, and separate estimates were calculated for respondents interviewed directly and surrogate respondents. We used equal care in all types of interviews and in all subject areas covered in the interviews; however, the data we obtained in interviews with surrogates still differed somewhat from those obtained in direct interviews. Inaccurate reporting of exposure tends to bias odds ratios toward the null value unless a systematic bias is present. Data from surrogate respondents are likely to introduce random error because of the surrogate's lack of detailed knowledge of the subject's exposure. On the other hand, it is possible that the surrogates for patients with lung cancer might tend to underreport the exposure contributed by their own smoking to a greater extent than surrogates for control subjects. Such a difference could mask an actual increase in risk or reverse the direction of the association. The findings shown in Table 3 indicate that the use of data from surrogates may have led to an underestimation of the effect of exposure from the spouse. Although our results for exposure due to smoking by the spouse differ from those of earlier studies,[18] our findings regarding other types of household exposure support the conclusion that exposure to environmental tobacco smoke can cause lung cancer.

Akiba et al.,[8] Dalager et al.,[9] and Garfinkel[3] have reported elevations in risk of 30 percent, 50 percent, and 10 percent, respectively, associated with exposure to a spouse's smoking; none of these increases were statistically significant. With the exception of Chan et al.[24] and Koo et al.[25] in Hong Kong, these and most other investigators have reported point estimates that suggest an increased risk for those exposed. The duration of exposure, as measured by the number of years the spouse smoked while living with the subject, did not have a statistically significant effect in our data. Two studies that used the same measure

2023382400

Source: https://www.industrydocuments.ucsf.edu/docs/zkyx0000

of exposure also failed to exclude the null value.[8,9] Garfinkel et al.,[7] using a different measure for duration of exposure (husband's smoking in the last 5 and 25 years), found one significant association among the large number examined. Exposure due to smoking by the spouse, expressed in terms of pack-years while the spouse was living with the subject, was found not to be significantly associated with lung cancer. Using a comparable measure of exposure, Trichopoulos et al.[5] reported relatively large increases in risk (greater than twofold). Perhaps our data do not show that smoking by the spouse increased the risk by itself because smoking by the spouse made up only about one third of the subjects' lifetime exposure to environmental tobacco smoke. It is also possible that physical circumstances and differences in study areas, the size of residences, ventilation, and other important physical aspects of the living conditions, as well as social habits that affect exposure within the family, will need to be measured and analyzed before the differences in findings among the studies can be reconciled.

The evidence we report lends further support to the observation that passive smoking may increase the risk of subsequent lung cancer, and it suggests that it may be particularly important to protect children and adolescents from this environmental hazard.

We are indebted to Andreas Nicolaou for his assistance with the computer programming used in our analyses.

References

1. Department of Health, Education, and Welfare. The health consequences of smoking: a report of the Surgeon General: 1972. Washington, D.C.: Government Printing Office, 1972:121-35. (DHEW publication no. (HSM)72-7516.)
2. Department of Health and Human Services. The health consequences of involuntary smoking: a report of the Surgeon General. Washington, D.C.: Government Printing Office, 1986. (Publication no. DHHS (CDC) 87-8398.)
3. Garfinkel L. Time trends in lung cancer mortality among nonsmokers and a note on passive smoking. J Natl Cancer Inst 1981; 66:1061-6.
4. Hirayama T. Cancer mortality in nonsmoking women with smoking husbands based on a large-scale cohort study in Japan. Prev Med 1984; 13:680-90.
5. Trichopoulos D, Kalandidi A, Sparros L. Lung cancer and passive smoking: conclusion of Greek study. Lancet 1983; 2:677-80.
6. Correa P, Pickle LW, Fontham E, Lin Y, Haenszel W. Passive smoking and lung cancer. Lancet 1983; 2:595-7.
7. Garfinkel L, Auerbach O, Joubert L. Involuntary smoking and lung cancer: a case-control study. J Natl Cancer Inst 1985; 75:463-9.
8. Akiba S, Kato H, Blot WJ. Passive smoking and lung cancer among Japanese women. Cancer Res 1986; 46:4804-7.
9. Dalager NA, Pickle LW, Mason TJ, et al. The relation of passive smoking to lung cancer. Cancer Res 1986; 46:4808-11.
10. Kabat GC, Wynder EL. Lung cancer in non-smokers. Cancer 1984; 53:1214-21.
11. Sandler DP, Wilcox AJ, Everson RB. Cumulative effects of lifetime passive smoking on cancer risk. Lancet 1985; 1:312-5.
12. Sandler DP, Everson RB, Wilcox AJ, Browder JP. Cancer risk in adulthood from early life exposure to parents' smoking. Am J Public Health 1985; 75:487-92.
13. Sandler DP, Everson RB, Wilcox AJ. Passive smoking in adulthood and cancer risk. Am J Epidemiol 1985; 121:37-48.
14. Pershagen G, Hrubec Z, Svensson C. Passive smoking and lung cancer in Swedish women. Am J Epidemiol 1987; 125:17-24.
15. Koo LC, Ho JH, Saw D, Ho CY. Measurement of passive smoking and estimates of lung cancer risk among non-smoking Chinese females. Int J Cancer 1987; 39:162-9.
16. Humble CG, Samet JM, Pathak DR. Marriage to a smoker and lung cancer risk. Am J Public Health 1987; 77:598-602.
17. Chan WC, Fung SC. Lung cancer in nonsmokers in Hong Kong. In: Grundmann E, Clemmesen J, Muir C, eds. Geographical pathology in cancer epidemiology. New York: Gustav Fisher Verlag, 1982:199-202.
18. Wald NJ, Nanchahal K, Thompson SG, Cuckle HS. Does breathing other people's tobacco smoke cause lung cancer? BMJ 1986; 293:1217-22.
19. Varela LR. Assessment of the association between passive smoking and lung cancer. (Ph.D. dissertation. New Haven, Conn.: Yale University, 1987.)
20. Breslow NE, Day NE. Statistical methods in cancer research. Vol. I. The analysis of case-control studies. Lyon, France: International Agency for Research on Cancer, 1980. (IARC scientific publications no. 32.)
21. Maclure M, Katz RB, Bryant MS, Skipper PL, Tannenbaum SR. Elevated blood levels of carcinogens in passive smokers. Am J Public Health 1989; 79:1381-4.
22. Cummings KM, Markello SJ, Mahoney MC, Marshall JR. Measurement of lifetime exposure to passive smoke. Am J Epidemiol 1989; 130:122-32.
23. Coultas DB, Peake GT, Samet JM. Questionnaire assessment of lifetime and recent exposure to environmental tobacco smoke. Am J Epidemiol 1989; 130:338-47.
24. Chan WC, Colbourne MJ, Fung SC, Ho HC. Bronchial cancer in Hong Kong 1976-1979. Br J Cancer 1979; 39:182-92.
25. Koo LC, Ho JH-C, Saw D. Active smoking and passive smoking among female lung cancer patients and controls in Hong Kong. J Exp Clin Cancer Res 1983; 42:367-75.

Massachusetts Medical Society
Registry on Continuing Medical Education

To obtain information on continuing medical education courses in the New England area, call between 9:00 a.m. and 12:00 noon, Monday through Friday, (617) 893-4610 or in Massachusetts 1-800-322-2303, ext. 1342. If writing, direct correspondence to: Program Registrar, Massachusetts Medical Society, 1440 Main St., Waltham, MA 02154-1649. The booklet is free to MMS members, $5.00 for nonmembers.

2023382401

Source: https://www.industrydocuments.ucsf.edu/docs/zkyx0000